lapsed into coma and ceased spontaneous respirations, requiring mechanical support for the following 48 hours. Since alkalosis shifts the hemoglobin-oxygen saturation curve in the same direction as does 2,3 DPG deficiency (that is, to the "left"), the effects of bicarbonate and hypophosphatemia (and therefore 2,3 DPG deficiency) were additively, and crucially, deleterious to this patient.

Analogous considerations have suggested to Dr. Carroll Leevy that some confusional states noted in malnourished alcoholic patients may also reflect red cell 2,3 DPG deficiency with resulting cerebral hypoxia. Administration of phosphate to some such patients has improved their sensorium, while concomitantly increasing red cell 2,3 DPG and decreasing spinal fluid pyruvate and lactate levels (discussion in reference 1).

These observations indicate clearly that administration of alkali to hypophosphatemic persons may be particularly hazardous. Patients with diabetic, or uremic, acidosis are potential victims of such well-intentioned maneuvers. Thus, the acidotic diabetic patient upon entry to hospital frequently manifests diminished red cell 2,3 DPG levels. This deficiency results mainly from the inhibitory effect of acidosis on red cell glycolysis (and hence 2,3 DPG synthesis). The deficiency worsens if hypophosphatemia supervenes, as it often does, during insulin therapy. Fortunately, the predicted inefficient release of oxygen by hemoglobin in this situation is ameliorated by the opposing beneficial effect on oxygen unloading of acidosis itself (the Bohr effect). Rapid correction of acidosis by administration of alkali reverses this ameliorating effect which may partially account for the not-infrequent disasters encountered with bicarbonate therapy. Similarly, uremic patients are frequently treated with antacid chelators to reduce serum phosphate levels and thereby inhibit secondary hyperparathyroidism stimulated by hyperphosphatemia. Such therapy is frequently overly-vigorous so that patients actually become hypophosphatemic. Rapid correction of acidosis, especially by hemodialysis in these patients, may lead to tissue hypoxia—a sequence which probably, at least partially, explains the postdialysis syndrome of lethargy and weakness noted in many such patients.

It should be evident from the above and from Dr. Fitzgerald's excellent review, that serum phosphorus homeostasis is a critical area for future investigation. For the present, rigorous maintenance of serum phosphorus should prevent many of the previously-unperceived, deleterious consequences associated with its deficiency.

HARRY S. JACOB, MD
Professor, Department of Medicine
University of Minnesota Medical School, Minneapolis

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Federal Support for Assumptions or for Science?

IN HIS PRESIDENTIAL ADDRESS to the Western Society for Clinical Investigation (which appears elsewhere in this issue), Dean Mason calls attention to a matter which should be of enormous concern to both the medical profession and the public. This is the substantial shift of dollar support away from basic research in medical science, which leads to new discoveries and fundamental progress, to programs which are expected to fill some presumed or assumed need for specific research to solve some specific unknown which will have significant impact in patient care. Further, substantial federal dollar support has been diverted entirely from support of medical research and is being used to impose costly yet untested assumptions on the health care delivery system. For example, there has been very little real testing of the assumptions inherent in Health Maintenance Organizations (HMO's), Professional Standards Review Organizations (PSRO's), the Health Services Agencies (HSA's) or of the approach of the Federal Drug Administration (FDA).

In retrospect this use of federal dollars to bring about an identified scientific or social purpose seems to stem from experience with the Manhattan Project during World War II. Here an enormous number of federal dollars was poured into a scientific project with a social (in this case military) purpose with an enormous and very tangible result. Now we seem to be trying for another Manhattan Project in health care delivery. The thing that appears to be forgotten is that without the basic scientific knowledge, a Manhattan Project type of approach will not succeed. The idea that if enough dollars are spent any assumption can be made to work is simply fallacious. Yet this is what we seem to be doing, and at the expense of federal support of the basic research which is the true well-spring of both scientific and social progress in health care and in the quality of life. As Mason says, this needs somehow to be brought to the public consciousness.

—MSMW

CORRECTION

In the case report "Cardiomegaly—A Possible Factor in Permanent Endocardial Pacing," which appeared in the April 1975 issue of THE WESTERN JOURNAL OF MEDICINE, the two photographs in Figure 2 (page 333) were exchanged inadvertently. They appear below as they should have been printed in the article.

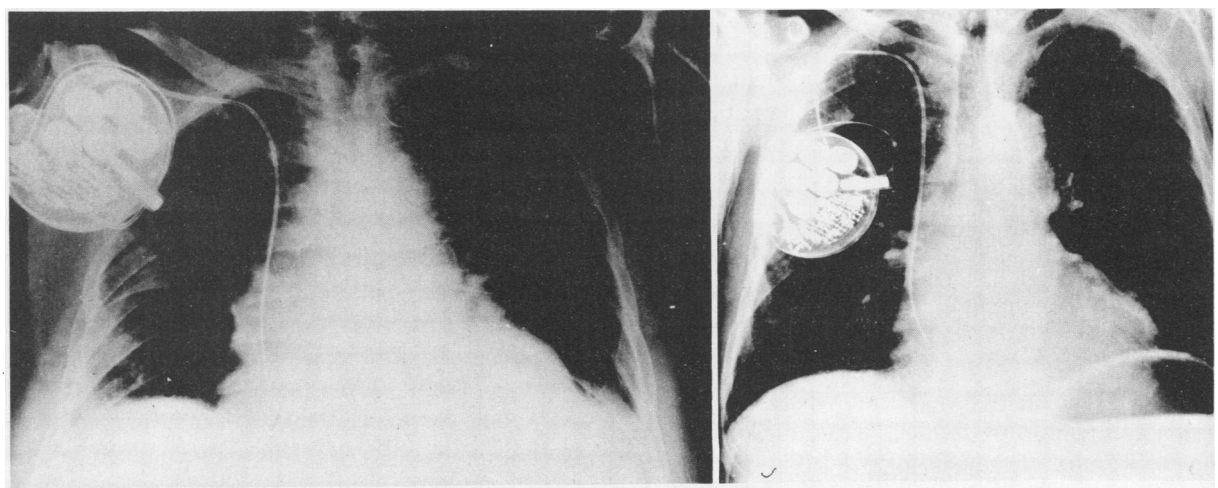

Figure 2.—(Case 2) **Left,** Decompensated cardiomegaly with transient exit block. **Right,** Compensated cardiomegaly with capture.